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INTRODUCTION

A recent study has defined a period of transient hyperexcitability in laryngeal adductor neurons in beagle pups. This suggests that age-related neurological instability of laryngeal mechanisms and abnormal laryngeal closure may be a cause of transient upper airway obstruction inducing potentially fatal central apnea, resulting in the Sudden Infant Death Syndrome (SIDS).[1] Furthermore, an increase in temperature has been implicated in sudden and unexpected infant death.[2] Therefore, the purpose of this investigation is to study the effect of temperature on the laryngeal adductor reflex, which has been shown to be of possible etiologic significance in SIDS.

SIDS is the most common cause of postneonatal death in the first year of life, with an incidence of 2 to 3 per 1,000 live births and an estimated mortality of 10,000 annually in the United States.[3,4] Although numerous theories have been proposed[5] to explain the sudden and unexpected nature of infant death, most theories fail to accommodate its unique epidemiologic and pathologic features. Such epidemiologic features include: 1) a peak incidence at age two to four months, sparing those younger than two weeks and older than one year of age, 2) death during sleep, 4) a familial aggregation of cases, and 5) symptoms of a mild upper respiratory tract infection during the two weeks before death in half the SIDS victims.[4,5,6]

Significant pathologic features of SIDS include: 1) intrathoracic petechiae, 2) media hypertrophy of small pulmonary arteries, 3) mild right ventricular hypertrophy, 4) retained brown fat, and 5) extramedullary hematopoiesis.[7,8,9,10] The last four pathologic features are thought to be caused by chronic systemic hypoxemia which is, in turn, attributed to chronic alveolar hypoxia and chronic alveolar hypoventilation, since there is no evidence of congenital cardiovascular anomalies or other disorders which may increase pulmonary vascular resistance and produce the above mentioned pathology. However, chronic alveolar hypoventilation is not caused by primary pulmonary pathology because the lungs of SIDS victims have not yielded significant findings at autopsy.[11] Therefore, recent efforts have been expended on theories which are compatible with the structural abnormalities of SIDS; theories which account for chronic alveolar hypoventilation in the absence of pulmonary pathology. These theories narrow to include: 1) central apnea and 2) upper airway obstruction.

Chronic alveolar hypoventilation may be the result of an altered and unstable respiratory pattern during sleep. Decreased ventilatory response to CO_2 in sleep is known to occur in the normal adult.[12] In normal sleeping neonates, lowered arterial oxygen tension[13] as well as periodic breathing and apnea of short duration have been noted.[14] More important, during sleep, infants with aborted SIDS, when compared with normal controls, have a higher incidence of periodic breathing and apnea,[15] prolonged apnea,[16] an abnormal ventilatory response to CO_2,[17] and alveolar hypoventilation.[16] The change in respiratory pattern in the normal sleeping neonate seems to be even more exaggerated in the

infant with aborted SIDS. An altered and unstable respiratory pattern may predispose these infants to prolonged and fatal apnea. Observations of infants with aborted SIDS, some of whom have had prolonged apneic episodes which necessitated resuscitation and some of whom have subsequently succumbed to SIDS, led Steinschneider to propose that apnea is the common pathway of death.[19]

Upper airway obstruction is thought to be another possible cause of chronic alveolar hypoventilation. Indeed, alveolar hypoventilation and insensitivity to hypercarbia occur in children with chronic upper airway obstruction,[20,21] as in infants with aborted SIDS. Furthermore, high negative intrathoracic pressure which occurs during upper airway obstruction is thought to produce intrathoracic petechiae, a consistent anatomical marker of SIDS.[5] Thus, upper airway obstruction is hypothesized as an important etiologic mechanism.[4,5]

Of interest, transient upper airway obstruction has been found to induce central apnea suggesting that the two theories may not be mutually exclusive. In sleep recordings,[22,23] premature infants and infants with aborted SIDS continue to make respiratory efforts transiently when nasal thermistors record an absence of airflow, indicating upper airway obstruction. This brief phase is then followed by complete cessation of thoracic movement and diaphragmatic activity, indicating a progression to central apnea. Thus these infants demonstrate upper airway obstruction preceeding and possibly inducing central apnea. This is a reason for the frequent failure of conventional apnea alarms which measure only thoracic movements

to detect these apneic episodes.[24] In support of transient upper airway obstruction inducing central apnea as an etiologic mechanism, neither abrupt and sustained tracheal occlusion nor hypoxia alone have been shown to produce significant intrathoracic petechiae in rats. Transient tracheal occlusion, vigorous respiratory efforts and a period of hypoxia prior to cardiac arrest have been identified to increase the incidence of this structural abnormality.[25]

These observations have prompted consideration of the possible role of laryngeal mechanisms in causing upper airway obstruction as a trigger to central apnea. It has been known that stimulation of the superior laryngeal nerve (SLN) produces both laryngospasm[26] and phrenic inhibition.[27] Laryngeal stimulation in newborn piglets produces prolonged apnea and asphyxial death.[28] Similarly, SLN stimulation in infant monkeys evokes transient laryngeal closure and prolonged apnea which continues after stimulation has ceased. This phenomenon is not observed in the adult monkey.[29]

Recent neurophysiologic studies have further clarified the age-dependent nature of the laryngeal closure reflex. Sasaki has defined a period of transient hyperexcitability of the laryngeal adductor nucleus in pups fifty to seventy-five days of age.[1] Bilateral train stimuli applied to the SLN's evokes action potentials consistent with laryngospasm in this age group. This is attributed to progressive neuronal maturation, and a transient decrease in both central latency and central inhibition, reflecting a differential maturation of excitatory and inhibitory influences on the reflex. These observations accommodate an important

epidemiologic feature of SIDS, that it occurs not immediately after birth but at a defined period thereafter, prior to complete neurologic maturation.

Sasaki proposes that transient adductor hyperexcitability, an age-related functional imbalance, may produce a precarious state of neurologic instability. Modified by sleep and other factors, such instability may result in respiratory disruption of sufficient severity to result in SIDS.

In support of this hypothesis, the excitability of other reflexes have also been found to be age-related and modified by sleep. Before two weeks of age, both the monosynaptic masseteric reflex and the polysynaptic digastric reflex in the kitten are greatest during rapid eye movement (REM) sleep and least while awake.[30] Apnea most commonly occurred in REM sleep in infants with aborted SIDS and some future SIDS victims.[19] The monosynaptic hindlimb reflex in the kitten develops in a parallel course.[31]

The sleep state is an important factor modifying the adductor reflex. Mechanical and chemical laryngeal stimulation in the adult dog induce prolonged apnea during REM sleep but not in non-REM (NREM) sleep or wakefulness.[32] This suggests that laryngeal mechanisms which induces apnea may be more active during REM sleep. In the neonatal lamb, laryngeal adductor activity is found to be reduced or absent during REM sleep but is increased and sustained during episodes of spontaneous apnea. This is not observed in the fetal or adult sheep[33] suggesting that laryngeal-induced central apnea (LICA) during REM sleep is age-related.

Age-related risk and sleep state are important factors in the study of etiologic mechanisms in SIDS. To this group of factors, temperature has recently been added as an important factor in sudden and unexpected death in infants.[2]

The control range, the range of environmental temperature at which body temperature can be kept constant, is more limited in the infant than in the adult.[34] Thus, beyond the upper limit of this narrow range, the infant becomes rapidly hyperthermic.[34,35] For example, a 2°C increase in incubator temperature may result in a naked infant becoming febrile.[36] A limited ability to thermoregulate holds true for other young mammals. The rectal temperature of kittens increases rapidly with thermal challenge compared to that of adult cats which tends to stabilize after a small increase.[37]

A narrow limit of thermoregulation in infants makes them more susceptible to hyperthermia in the face of thermal challenge. SIDS victims may also have been predisposed to body temperature changes as a prospective study found in them a higher incidence of spontaneous hyperthermia and hypothermia.[38]

The rise in both incubator and skin temperature have been associated with higher incidences of apnea.[39,40] Similarly, cessation of respiration due to warming of newborn infants[41] and lambs[41,42] has been observed. Furthermore, temperature increases has been implicated in sudden and unexpected deaths in infants who have had strong clinical evidence of overheating and some pathologic evidence of heat stroke.[2] Thus, SIDS victims who have been more likely to experience hyperthermia in response to thermal challenge may be more likely to succumb to LICA.

REM sleep may be another factor which places the infant at risk for hyperthermia and therefore LICA. During REM sleep, sweating is inhibited[43] while the rate of metabolism increases.[44] Although studies of thermoregualtion of the infant during sleep are unavailable, both the skin and rectal temperatures of the adult show rhythmic increases synchronized with REM sleep and the cessation of sweating, only to decrease again at the end of this sleep state. In warmer conditions, the basal downward trend of body temperature during the night is abolished but the phasic fluctuations during REM sleep persist.[43]

It is of interest that during REM sleep, a time when an increase in body temperature occurs, the incidence of apneic episodes in infants with aborted SIDS is higher. Furthermore, most SIDS deaths occur during sleep. This gives credence to the theory that REM sleep in conjunction with a limited thermoregulatory ability and increased hyperthermia, predisposes an infant to hyperthermia which may cause LICA.

In a previous neurophysiologic study, a transient period of laryngeal adductor neuronal hyperexcitability has been defined. This precarious state of neurologic instability modified by temperature, REM sleep and other factors known to be related to SIDS may produce LICA. The purpose of this investigation is to study the influence of body temperature on the laryngeal closure reflex, to discover whether this reflex is further exaggerated by temperature changes during its period of hyperexcitability.

Temperature, REM sleep, an age-related incidence are modifying factors of an etiologic mechanism in SIDS. Yet the interrelationship of the individual factors is complex. Hyperthermia is found in infants during

thermal challenge, occurs in REM sleep, and causes high incidences of apnea. The incidence of spontaneous hyperthermia is increased in SIDS victims. REM sleep makes up a larger fraction of the day of a newborn,[45] is associated with body and skin temperature increases, and claims a higher incidence of apnea than NREM sleep. Laryngeal mechanisms may induce central apnea during REM sleep in animals of a specific age.

Among these complex interrelationships, the effect of body temperature on age-dependent laryngeal mechanisms has not been studied. Filling this gap in our knowledge will contribute to an understanding of laryngeal neurophysiology and of etiologic mechanisms causing death in SIDS.

MATERIALS AND METHODS

Seventeen beagle pups in three age groups---3 weeks, 6 weeks and 12 weeks, and eight adult dogs were anesthetized with pentobarbital sodium 30 mg per kg, intraperitoneally in puppies and intravenously in adults. Anesthesia was kept at Stage III plane I. The right femoral artery or one of its branches was cannulated for arterial blood gas samples. The right femoral vein or one of its branches was cannulated for administration of phenobarbital.

The right SLN and right thyroarytenoid muscle (TAM) were exposed and prepared according to the method developed by Sasaki.[1,46] (Fig. 1) A midline neck incision was made from the level of the hyoid to the sternum. The trachea was exposed and a tracheostomy made at the fourth ring. A cannula was inserted for spontaneous respiration. The right strap muscles were identified and transected to expose the thyroid cartilage. The internal branch of the right SLN was identified under the operating microscope, carefully dissected and cut near its entrance to the thyroid cartilage. The nerve sheath was pealed in a mineral oil bath to prevent drying. The external branch was identified and transected.

The right thyroid ala was carefully dissected from the laryngeal muscles below without injuring branches of the recurrent laryngeal nerve (RLN). The inferior horn was disarticulated from the cricoid, and the thyroid ala was reflected to expose the TAM, the major adductor of the larynx.[47] The superior horn was transected in younger puppies to ensure adequate exposure. Hooked platinum-irridium electrodes were inserted into the TAM for recording action potentials. A bipolar platinum electrode was used for stimulation of the SLN.

Single-shock electrostimuli of 0.1 msec and 0.8 to 15 volts were delivered in rectangular waves to the central end of the right SLN by a Grass stimulator (S8) and isolation unit (4678). Each stimulus was phase locked into early expiration for data consistency because the threshold of the glottic closure reflex was found to increase on inspiration and to decrease on expiration.[48] Evoked responses from the ipsilateral TAM were amplified by a preamplifier (WP DAM 5A), displayed on an oscilloscope (Tektronics 5103N), recorded on an instrumental recorder (Hewlett Packard 3960) and photographed (Grass C4R). Rectal temperature was monitored continuously (tele-thermometer YSI 400). Arterial blood gas samples were analyzed for every 2^o to 3^o of body temperature change (Radiometer RHM-72). Values were maintained within normal physiologic limits--pH 7.35-7.45, $pO_2 > 80$mm and pCO_2 30-40mm. This was because the reflex threshold decreased with decreases in pO_2,[49] and synaptic transmission and membrane resistance were affected by changes in pH.

For each of the four age groups of dogs, the threshold and latency of the TAM were measured as a function of body temperature per degree from 34^o to 41^oC. Each parameter was measured from ten independent stimulations of the SLN. An average was taken and recorded.

Dogs were cooled with ice packs and warmed with an electric blanket.

Data Analysis

The Student's t test was used.

RESULTS

Stimulation of the right SLN produced action potentials from the ipsilateral TA. (Fig. 2-5).

The latency (Table I, Fig. 6, 8-11) was higher at lower temperatures and decreased linearly with increasing body temperature in dogs of all four age groups--3 week pups, 6 week pups, 12 week pups and adults. At a given body temperature, latency decreased with age, with but a few exceptions. The curves of the 12 week old and the adult were almost superimposed at the higher temperature ranges.

The threshold, like latency, was higher at the lower temperature range and decreased with increasing body temperature for all four age groups. (Table II, Fig. 7, 8-11) In all cases, the threshold decreased exponentially with increasing body temperature. The slope of the curves of the 3 and 6 week old puppies appeared steeper than those of the two older age groups. (Fig. 7) The slope of the curves of the two younger age groups seemed to be steepest at temperature 37^{0} to 39^{0}C which included the normal physiological range of body temperature. Small increases in temperature in this range produced substantial decreases in threshold.

The adductor reflex was inhanced at higher temperatures where both threshold and latency were decreased. This was especially true for the threshold of puppies at 3 and 6 weeks of age, and temperature range of 37^{0} to 39^{0}C.

Table I

Latency of adductor reflex in A) 3 week old, B) 6 week old, C) 12 week old and D) adult beagles, at 34 to 41 C. Latency in msec. ± S.D.

Temp C	A		C	D
34	22.0 ± 2.0	19.3 ± 2.0	19.0 ± 0.0	18.5 ± 0.5
35	21.2 ± 1.6	19.2 ± 1.6	16.5 ± 1.0	16.7 ± 1.1
36	20.3 ± 1.8	18.8 ± 1.5	15.9 ± 1.9	14.8 ± 1.8
37	19.5 ± 1.8	18.2 ± 1.6	14.6 ± 2.1	14.1 ± 1.7*
38	18.6 ± 1.8	16.8 ± 1.3	13.3 ± 1.8	14.0 ± 0.7
39	17.9 ± 1.8	15.8 ± 1.2	12.6 ± 2.1	13.0 ± 1.3
40	17.2 ± 1.7	15.3 ± 1.2	12.3 ± 2.8	12.5 ± 1.5
41	16.5 ± 1.6	14.8 ± 1.6	12.5 ± 2.3	13.5 ± 0.5

$p < 0.05$ except where indicated by *

* = $p > 0.05$

Table II

Threshold of the adductor reflex in A) 3 week old, B) 6 week old, C) 12 week old and D) adult beagles at 34 to 41 C. Threshold in volts ± S.D.

Temp C	A	B	C	D
34	2.6 ± 1.0	1.8 ± 0.2	0.46 ± 0.00	0.33 ± 0.01
35	2.3 ± 0.2	1.73 ± 0.04	0.48 ± 0.04	0.27 ± 0.04
36	2.2 ± 0.07	1.43 ± 0.18*	0.44 ± 0.05	0.27 ± 0.02
37	2.1 ± 0.03	1.4 ± 0.44	0.38 ± 0.03	0.27 ± 0.03
38	1.68 ± 0.43	0.92 ± 0.43	0.28 ± 0.02	0.19 ± 0.02
39	1.15 ± 0.15	0.68 ± 0.21	0.27 ± 0.05	0.18 ± 0.03
40	0.95 ± 0.07	0.43 ± 0.11	0.26 ± 0.06	0.14 ± 0.04
41	0.98 ± 0.06	0.40 ± 0.06	0.26 ± 0.02	0.12 ± 0.04

$p < 0.05$ except where indicated by *

* = $p > 0.05$

DISCUSSION

The results of this study indicate that the latency and the threshold of the laryngeal closure reflex decrease as body temperature increases, and increase with fall in body temperature.

The reflex latency may be affected by the velocity of axonal impulse transmission and of synaptic transmission. It is known that the axonal conduction velocity increases with temperature increase.[51,52] The conduction velocity of an axon is the speed at which the action potential is propagated. The action potential is composed of a unique sequence of electrical changes which are themselves dependent upon the ionic conductance changes produced by depolarization. In terms of the Hodgekin-Huxley equations, the rate constants for Na+ and K+ conductance increase three times for every 10^{0}C rise in temperature.[53] A study of recorded action potentials reveal that the duration of each of its phases--depolarization, potential reversal, repolarization and hyperpolarization occurs more rapidly with temperature increase.[54] This contributes to a lowered latency.

At the neuromuscular junction, neuromuscular transmission is found to be slower with temperature decrease because of delayed and reduced release of neurotransmitter substance. Furthermore, neurotransmitters are not released in a concentrated burst as was the case at higher temperatures but are dispersed over a longer period of time.[55,56] The change in time course and quantity of neurotransmitter release contributes to a lengthened latency at a lower temperature.

Although it appears that the effect of temperature on the speed of synaptic transmission has not been studied carefully,[57] it is hypothesized

that chemical transmission at the synapse may also be affected by changes in temperature since its presynaptic events are thought to be similar to those at the neuromuscular junction. The neuromuscular junction has also been used as a model in the study of CNS synaptic transmission.[58] Thus, synaptic transmission may also be more rapid at a higher temperature and slower at a lower temperature.

An important consideration is whether the reduced and delayed neurotransmitter release may affect the threshold, whether a stronger stimulus is needed to effect an increased and sufficient amount of neurotransmitters as to generate an action potential at a lower temperature. At the neuromuscular junction, 200 to 300 vesicles of acetylcholine are released after an impulse arrives. This amount of neurotransmitters creates an endplate current flow about 3 to 4 times that required to stimulate the muscle. Therefore, the normal, disease free, neuromuscular junction is said to have a high "safety factor."[59] It is unlikely that the response at the motor endplate will be decreased by some reduction of neurotransmitters release produced by cooling. In short, the threshold is unlikely to be affected under these conditions. Both Katz[56] and Takeuchi[55] have shown that cooling produced a decrease in the number of responses at the motor endplate only in a neuromuscular preparation with a high Mg++ and low Ca++ concentration. This suggests that the threshold will only be affected by cooling under extreme experimental conditions.

On the other hand, synaptic transmission in the CNS does not have the high "safety factor" such that the threshold of a neuron may be

affected by temperature changes. As each action potential arrives at the synaptic knob or terminal, only one or more vesicles of neurotransmitters are emptied into the synaptic cleft. The average is one vesicle.[59] This amount of neurotransmitters usually produces an EPSP of less than one millivolt, a fraction of the 10 or more millivolts required for the generation of an action potential. An increase in membrane potential of this magnitude requires a simultaneous discharge of 10 to several hundred or more excitatory synaptic knobs.[59]

Although it appears that studies of the effect of temperature on CNS neurotransmitter release are unavailable,[57] neuronal threshold in the spinal cord is found to decrease with temperature increase.[52] This suggests that CNS synaptic transmission could be affected by temperature. Peripherally, the amount of neurotransmitters is shown to decrease ten times with a 19^0C decrease in temperature at a sympathetic ganglion.[60] Thus, it seems possible that a synaptic knob in the CNS which only releases one or more vesicle per impulse, may release fewer vesicles to none at all with cooling. Such an effect on all the excitatory synaptic knobs may result in the absence of action potential generation postsynaptically.

Neurons in the CNS are dependent for excitation on synaptic knobs which have a more limited capacity for neurotransmitter release, compared with the axon terminal at the neuromuscular junction. It is hypothesized that action potential generation in the CNS may be more sensitive to temperature changes such that with cooling, a stimulus of higher intensity may be required, resulting in a threshold that is raised. Similarly,

it is also hypothesized that warming may increase the number of vesicles released for each impulse which arrives at the synaptic knob, such that action potentials may be generated with a weaker stimulus. Thus, the threshold is lowered. Some interneurons have been shown to increase the frequency of discharge with increases in temperature,[61] suggesting that subthreshold stimuli may generate action potentials at higher temperature because of increase neurotransmitter release. Similarly, neurons which increase the rate of firing with increase in temperature have been found in the midbrain and the spinal cord.[62]

A change in the resting membrane potention (RMP) may affect the threshold. As defined by the Goldman equation,* the RMP is a function of temperature and ion conductance which are also temperature dependent.[51] To date, most studies have found little or no change in the RMP over a wide range of temperatures.[54,63] It is unlikely that such changes in the RMP could be the cause of the exponential change in threshold that occurs with temperature variation. Other studies have also shown that the threshold increases exponentially with temperature decrease and decreases in a similar fashion with temperature increase.[52,63]

$$* \quad EMP = \frac{R\,T}{F} \ln \frac{C_{1i} P_1 + C_{2i} P_2 + A_{3o} P_3 + A_{4o} P_4}{C_{1o} P_1 + C_{2o} P_2 + A_{3i} P_3 + A_{4i} P_4}$$

R = universal gas constant
T = absolute temperature
F = Faraday
C_i = concentration of cation inside the membrane
C_o = concentration of cation outside the membrane
A_i = concentration of anion inside the membrane
A_o = concentration of anion outside the membrane
P = permeability factor

The laryngeal adductor reflex is polysynaptic. Each neuron which is a component of this reflex has a particular threshold potential which must be attained for action potential generation.

There may be one or more neurons which define the reflex threshold, i.e. their threshold will only be attained with a stimulus at reflex threshold applied at the afferent limb. Other neurons in the chain may discharge with stimulus which is less than that required to attain reflex threshold. In the CNS, neurons which define the reflex threshold, may thus alter the reflex threshold with temperature changes. To test this hypothesis, more extensive peripheral and more important, central recordings will be necessary.

Other observations of this investigation are also in accord with those of previous studies. Sasaki[1] has found that the increase in conduction velocity of both the SLN and RLN occurred before 50 to 75 days of age in beagles. This is consistent with the findings in this study. The discrete period of hyperexcitability of adductor neurons, at 50 to 75 days of age, is not reproduced here because the bilateral, crossed adductor reflex has been studied in that particular investigation while the ipsilateral reflex is used here.

Nevertheless, this study shows that both the latency and the threshold of the laryngeal closure reflex decrease with hyperthemia. The effect of temperature on latency attributed to changes in axonal conduction and synaptic transmission velocity. Temperature dependent changes in synaptic transmission are also proposed to be the cause of threshold changes. Thus, temperature increase may enhance the reflex markedly during 50 to 75 days of age. This supports the hypothesis that increased body temperature

is one of the factors which could modify the adductor reflex, to enhance transient or abnormal laryngeal closure during its period of hyperexcitability. Our date therefore indicate that transient upper airway obstruction may in turn induce apnea of sufficient duration as to bring on sudden and unexpecteddeath in infants.

SUMMARY

A recent study has defined a period of transient hyperexcitability in laryngeal adductor neurons in beagle pups. This suggests that age-related neurological instability of laryngeal mechanisms and abnormal laryngeal closure may be a cause of transient upper airway obstruction inducing potentially fatal central apnea, resulting in the Sudden Infant Death Syndrome (SIDS). Temperature increases have been implicated in sudden and unexpected infant death.

This investigation has found the latency and the threshold of the laryngeal adductor reflex to decrease during hyperthermia. Thus, hyperthermia may enhance the reflex during its period of hyperexcitability to trigger upper airway induced central apnea of sufficient severity to result in death. The effect of temperature on latency is attributed to changes in axonal conduction and synaptic transmission velocities. Temperature dependent changes in synaptic transmission are hypothesized to be the cause of threshold changes.

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FIGURES AND LEGENDS

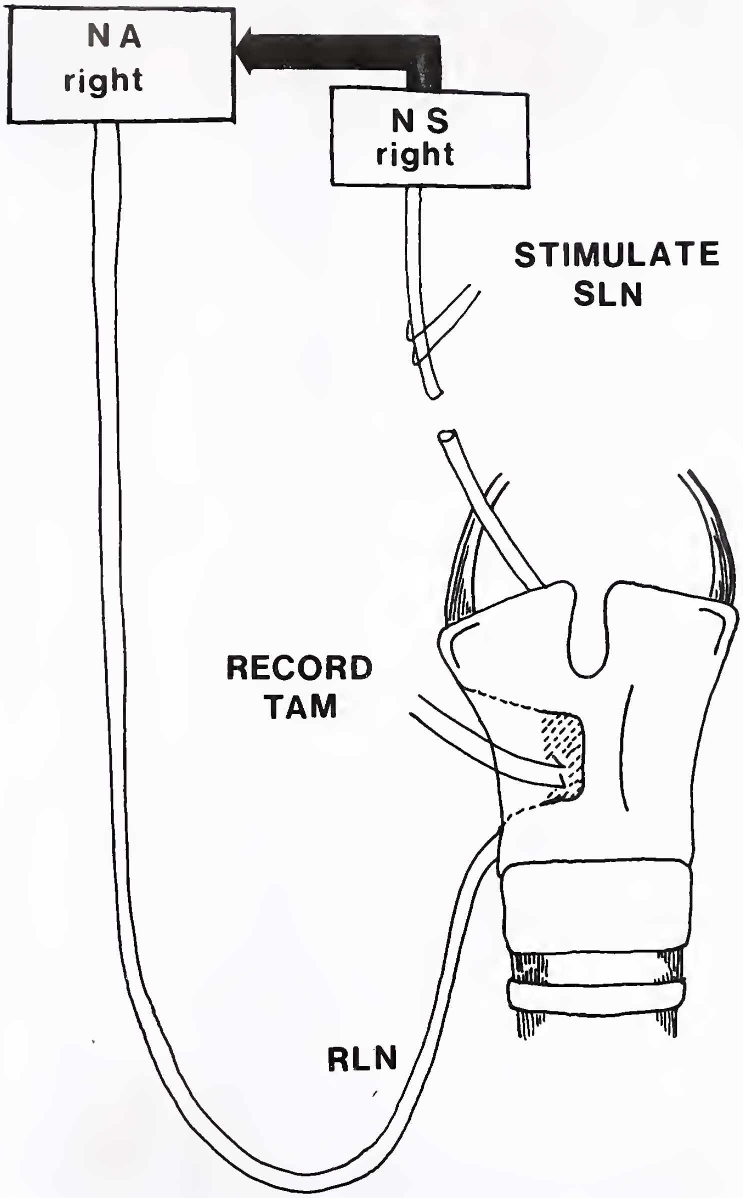

Fig. 1. The ipsilateral adductor evoked response is produced by stimulating the right SLN and recording from the right TAM. NS = nucleus solitarius, NA = nucleus ambiguus, SLN = superior laryngeal nerve, RLN = recurrent laryngeal nerve, TAM = thyroarytenoid muscle.

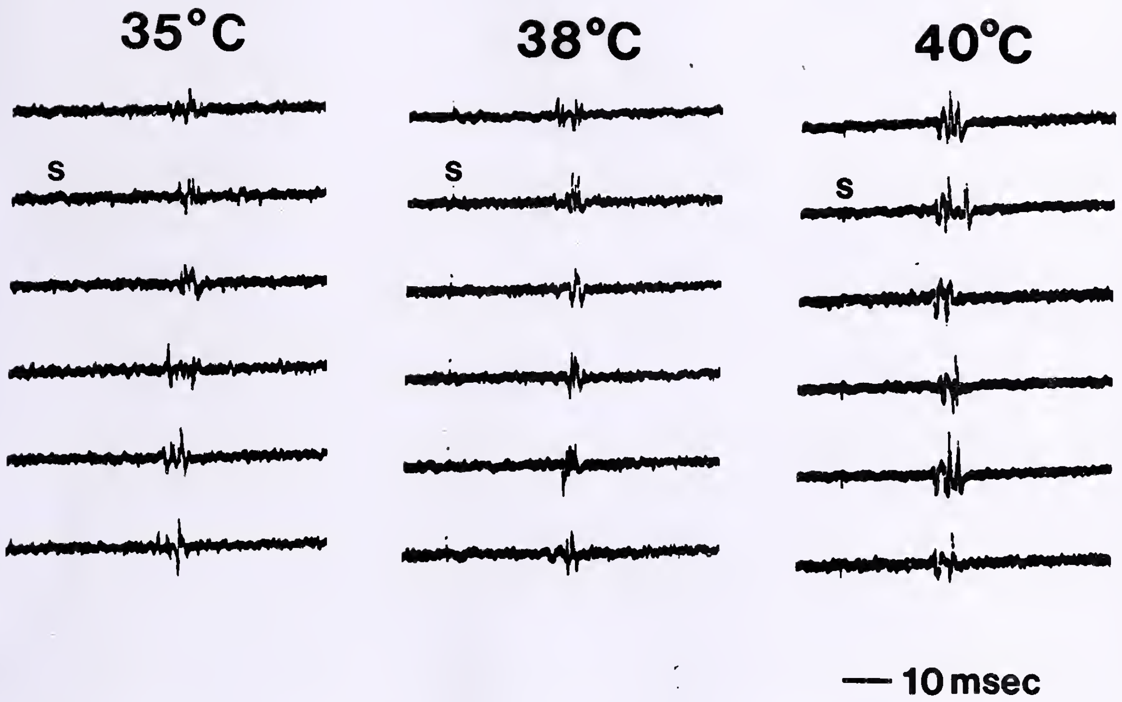

Fig. 2. Evoked adductor responses elicited by single-shock stimulation of SLN in 3 week old puppies. S denotes stimulus artifact.

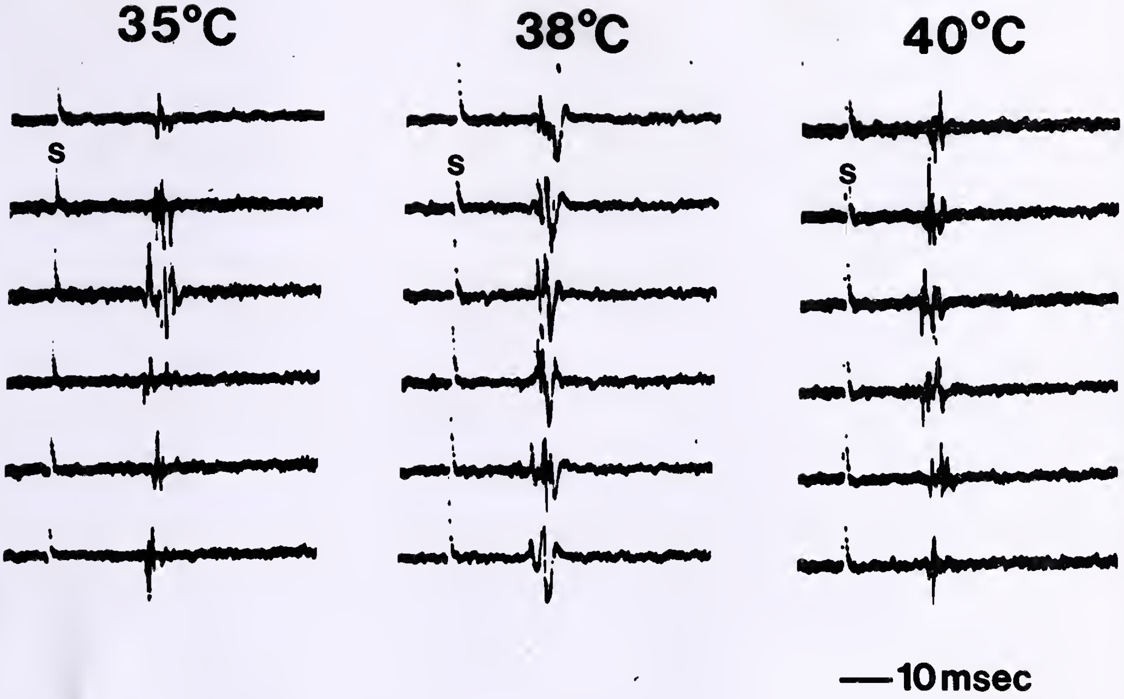

Fig. 3. Evoked adductor responses elicited by single-shock stimulation of SLN in 6 week old puppies.

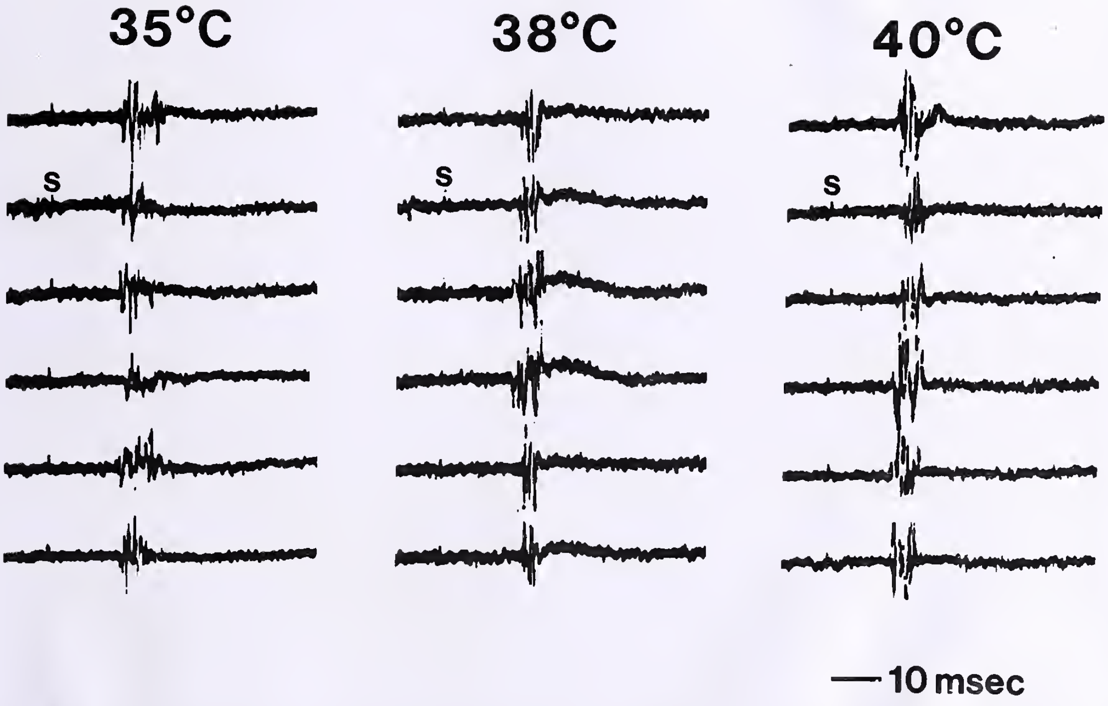

Fig. 4. Evoked adductor responses elicited by single-shock stimulation of SLN in 12 week old puppies.

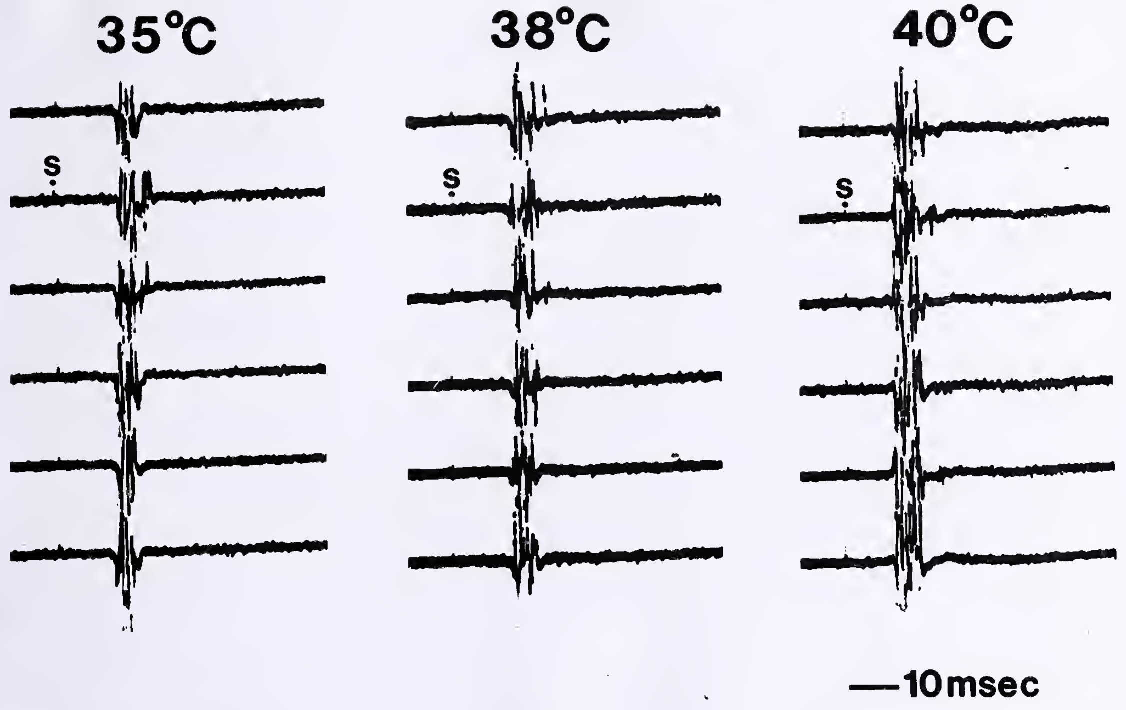

Fig. 5. Evoked adductor responses elicited by single-shock stimulation of SLN in adult dogs.

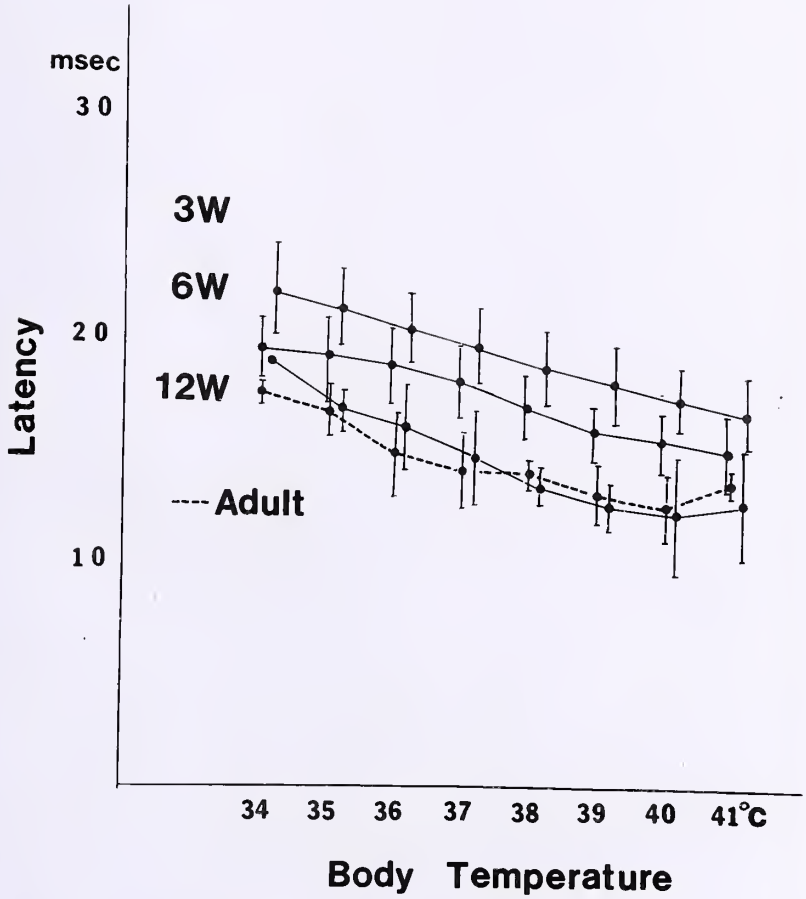

Fig. 6. The Influence of Body Temperature on Latency in Dogs in 4 age groups. Note the linear relationship in all Age Groups.

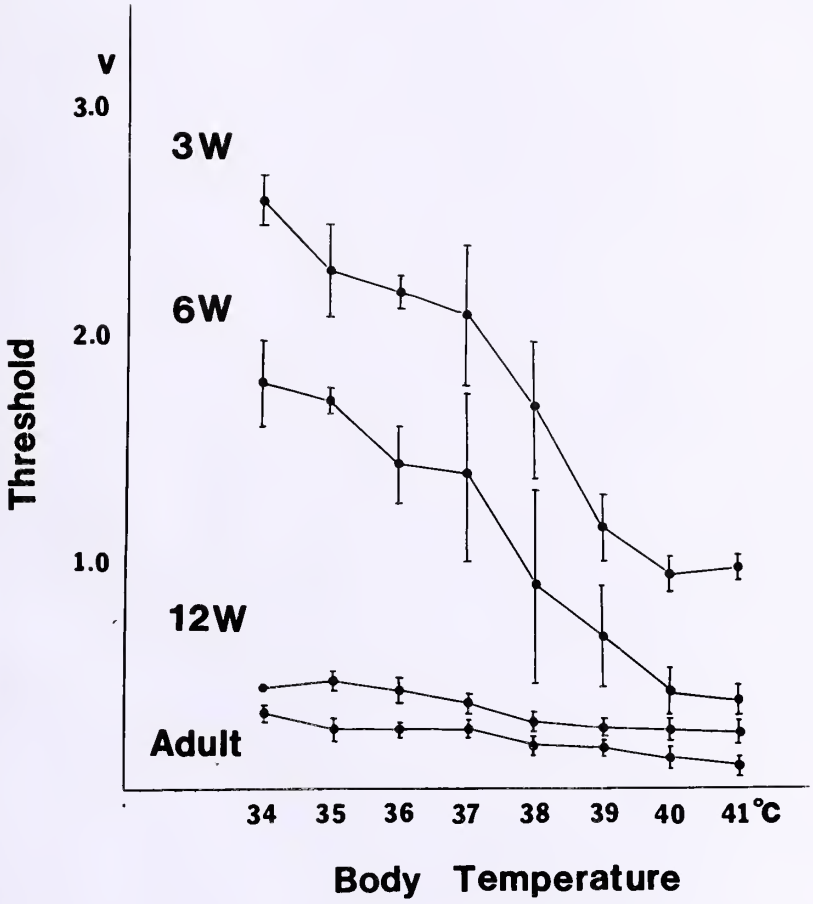

Fig. 7. The Influence of Body Temperature on Threshold in Dogs in 4 Age Groups.

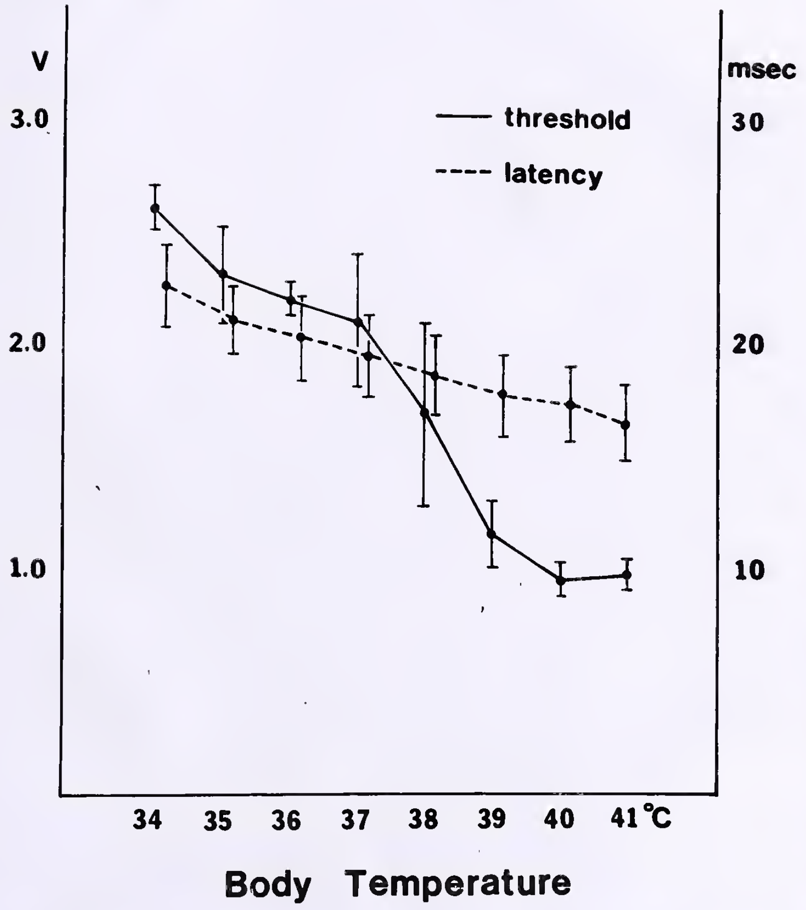

Fig. 8. The Influence of Body Temperature on Latency and Threshold in 3 week old Puppies.

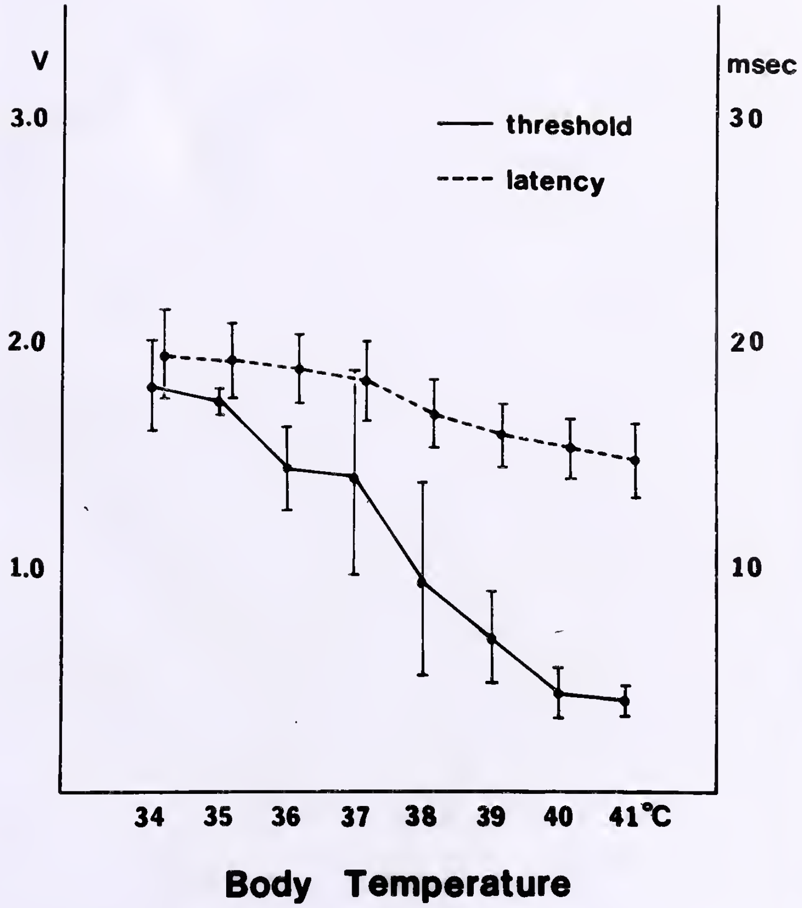

Fig. 9. The Influence of Body Temperature on Latency and Threshold in 6 week old Puppies.

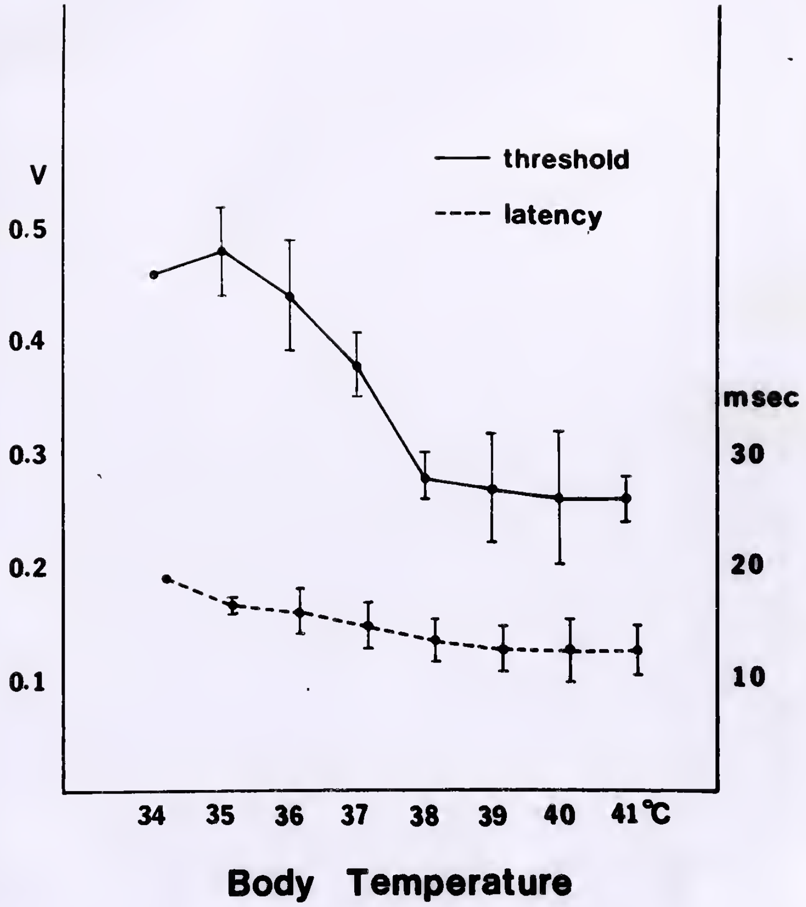

Fig. 10. The Influence of Body Temperature on Latency and Threshold in 12 week old Puppies.

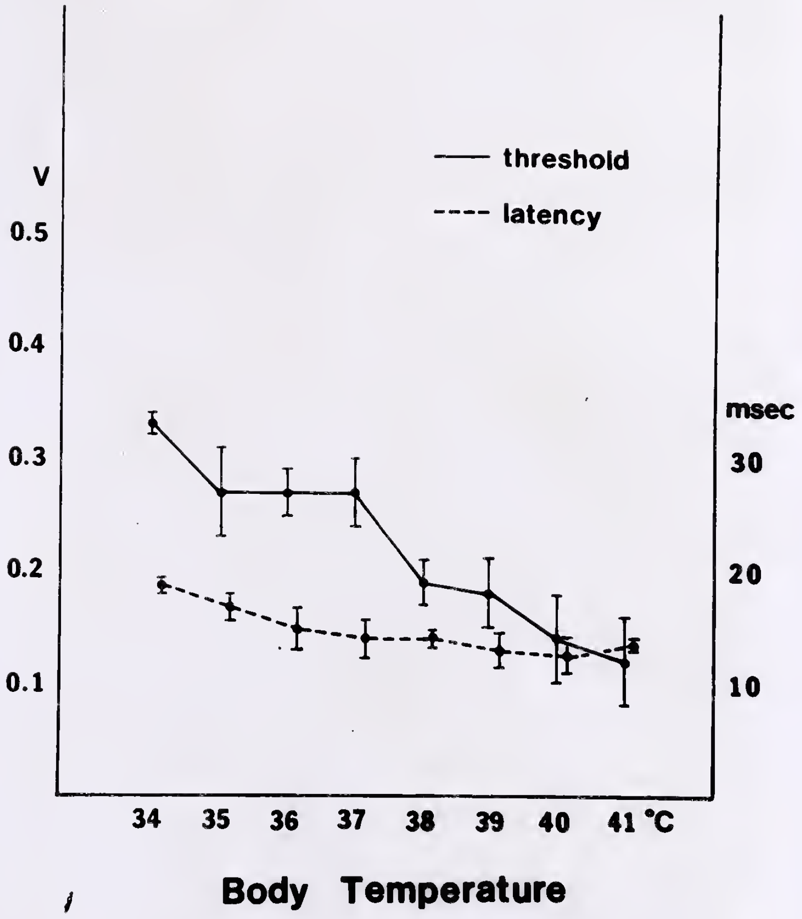

Fig. 11. The Influence of Body Temperature on Latency and Threshold in Adult Dogs.

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